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ON THE FORMS OF APHASIA MET WITH IN ABSCESS OF THE  
LEFT TEMPORAL LOBE.

BY

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An abscess in the left temporal lobe may give rise to a speech defect or it may not. Very considerable accumulations of pus may be situated in this region without any apparent speech disturbance, while, on the other hand, a small purulent focus may be enough to give rise to marked disturbance of speech. In speaking of defect of speech in this paper I intend to refer solely to aphasia, and not to either slowness of speech or to dysarthria.

Aphasia is probably present in nearly 50 per cent. of abscesses of the left temporal lobe. The exact form of aphasia met with is, however, rarely treated by writers on this subject with any degree of fulness or exactitude. The simple but vague statement that aphasia is present, being all that is found in the great majority of text-books, even in those dealing more especially with diseases of the nervous system. A few authors refer to *word-deafness* as being the only characteristic symptom of lesion in the temporal lobe. This form of aphasia is, however, comparatively rare. It would be a mistake to wait for its appearance in any case before concluding we had to do with an abscess in this situation. A pure and complete motor aphasia has been met with as also paraphasia, the presence of the former being explained by distant pressure, and that of the latter by the cutting off of the connections between the auditory and motor centres.

Within a few months I have had an opportunity of observing two cases of abscess consecutive to ear disease in the temporal lobe. In both a clinical type of aphasia was present which differs from the forms above referred to. The conditions were so similar that I believe they teach a useful lesson in helping to determine more easily what is often a difficult problem, *i. e.*, the differential diagnosis between temporal abscess and abscess elsewhere in the brain.

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\* Read before the Canadian Medical Association, Ottawa, September, 1900.

**CASE I.****Abscess of the Temporo-Sphenoidal Lobe Consequent on Purulent Otitis Media—Aphasia—Operation—Death Three Days after from Meningitis.**

A delicate man, aged 22, was admitted into the Royal Victoria Hospital on August 5, 1900, under the care of Dr. Buller. In the early part of March of the present year he had an attack of influenza followed by acute purulent otitis media. After suffering severely for four days from pain in the left ear, a discharge occurred, this being followed by great relief. Under treatment, the discharge gradually ceased and he was apparently well on the first of May, although deaf and suffering from tinnitus aurium. The treatment of the ear continued,—Politzer inflation twice weekly and painting the throat with an iodine-glycerine solution. This treatment was continued daily till the first of July and afterwards three times a week.

On the 15th of July there was a sudden and violent recurrence of the pain in the ear lasting for about ten days. On the 26th of July he was seized with rigors and vomiting, the pain and tinnitus at the same time ceasing. At that time his temperature ranged between 100° and 101.5° F. The headache recurred on many occasions, however, and at the time of his entrance into the hospital he was suffering very severely. The vomiting occurred two or three times daily and was distinctly cerebral in type.

On admission, he was found to be suffering from more or less constant and severe headache with vomiting. He was slow in answering questions and from the history it would appear that since the onset of the sudden pain, etc., ten days previously, he has had great difficulty in naming objects. This difficulty still persists. It is found that he is unable to mention the name of any object whatever. He does not know his own or his mother's name. He, however, has a very extensive vocabulary of words and knows their proper use. Although he is unable to name an object he has no trouble in proving that he understands the use of objects he is totally unable to name. He understands perfectly what is said to him and he also correctly obeys commands given in writing. He is able to write from dictation and to copy correctly. He reads correctly and easily. The left ear is not discharging; the canal is narrow. The membrana tympani is congested but not perforated. There is no swelling or tenderness about the mastoid; a watch is heard at half an inch. *Eyes.*—The pupils are normal in size and reaction. There is distinctly a beginning double optic neuritis.

A diagnosis of abscess of the temporo-sphenoidal lobe was made and immediate operation determined on. Dr. Buller opened into the

mastoid cells. He found pus. Dr. Bell proceeded to expose the brain over the temporo-sphenoidal region and after opening the dura, he found the parts bulging, but not pulsating.

With a trocar he was able without any trouble to find a pus-containing cavity in the left temporal lobe. Nearly three ounces were evacuated. The night after the operation the patient slept better than he had done for many weeks, being free from pain and discomfort. Even within twelve hours after the operation it was noticed by several observers that there was a distinct improvement in his speech. In twenty-four hours the improvement was so marked that it could be said that he had completely recovered his lost power. All kinds of test objects were promptly and correctly named. Unfortunately about thirty hours after the operation, symptoms of meningitis developed, which proved fatal forty-eight hours after their onset.

A postmortem examination was not obtained. This does not, however, detract much from the clinical value of the case, as during life the presence of the abscess had been demonstrated.

#### CASE II.

##### **Abscess of the Temporo-Sphenoidal Lobe Consequent on Purulent Otitis Media—Aphasia—Sudden Death from Respiratory Failure.**

A girl, aged 18, who spoke both French and English, was admitted into the Royal Victoria Hospital on the 16th of February, 1900, complaining of headache and vomiting. The headache dates back to Christmas of 1899. It has been persistently present and generally severe, keeping her awake at nights. Pain has been generally referred to the left temporal region until a few days ago, since when its site has been continuously frontal. The vomiting set in about four weeks after the pain in the head was felt. It has been, with the exception of the last few days, more or less persistent since its onset.

A difficulty in speaking has been noticed since the headache has been first complained of. She seemed unable to utter the word at once which she wished to say. One of her friends expressed the difficulty in the words that "she seemed for a few moments to have forgotten what she wanted to say and only by an effort would she say what she wanted." The difficulty of speech rapidly increased in severity and in the course of a few days reached the intensity that it presented at the time of her entrance to hospital, six months after its onset. During the past month she has had several general convulsive movements, lasting about fifteen seconds and attended by loss of consciousness.

*Past History.*—When nine months old the patient had a purulent discharge from each ear lasting for three months. At three years of

age she had pneumonia followed at intervals by measles, chicken pox and whooping cough. At fourteen she had a discharge from behind the right ear (eczema), lasting three months. For a period of three days in the month of October last she suffered from frontal headache and vomiting. Both ceased and she has remained perfectly well up to the onset of her present symptoms on Christmas day, 1899.

The family history is of no importance.

On admission, she was found to be in a lethargic state, answering even simple questions with difficulty. She speaks very slowly and takes a considerable time before she attempts the answer to a question. She is unable to tell even approximately the time of onset of her trouble. She does not misplace words. Although well educated, she is not able to comprehend written language fully, being only able to pick out in a sentence here and there short simple words. She is able to write her own name, but otherwise her writing to both dictation and copy is imperfect, especially so to dictation. When asked to write "main" she writes "yen." In copying "donnez" she writes "danny." When asked to name simple objects shown her, she is unable to do so. When shown a pen, a key, a watch, etc., she is quite unable to recall their names either in French or English. All spoken commands appear to be clearly and perfectly understood although the response is slow. She has greater difficulty in recognizing individual letters than individual words.

*Motor Power.*—There is a slight paresis of the lower respiratory branches of the right facial nerve. Movement in all other parts is normal.

*Sensation.*—There is neither subjective or objective disturbance in the face or extremities.

*Reflexes.*—Superficial normal; the knee jerks are much diminished and more so on the right than on the left side.

Dr. Buller reported on the conditions of the eyes and ears.

*Eyes.*—The right pupil is slightly larger than the left, but both react readily to light and accommodation. There is blurring of both optic discs and engorgement of the vessels, but it can hardly be said that there is a definitely established optic neuritis.

*Ears.*—She hears only on contact on both sides. There is a suppurative otitis media on the left side, with involvement of the bones of the roof of the tympanum. There is a profuse and fetid discharge from the left ear.

The patient remained in the state described for several days, the only noticeable addition to the above symptoms being the development of the optic neuritis.

A diagnosis of abscess of the temporo-sphenoidal bone was made and an operation was determined on, but a few hours before it was to have taken place, the patient suddenly expired, the immediate cause being respiratory failure. I am indebted to Dr. Shirres for the following notes of the postmortem examination performed by him.

The membranes situated in the left middle fossa of the skull were markedly thickened, particularly over that part of the petrosal bone which forms the roof of the tympanum. The bone beneath was carious. The tympanum was also found diseased, but the mastoid cells were normal. The brain in the region of the temporal lobe on the left side was distinctly swollen and oedematous, being at least a third larger than the lobe on the right side. The convolutions were flattened and sulci shallow. On attempting to remove the brain, an abscess, coming to within about 3 mm. of the surface, burst about a quarter of an inch below the Sylvian fissure with the escape of about 40 c.c. of pus, having a foul odour, a greenish tint, and an acid reaction. About the middle of the under surface of the third temporo-sphenoidal convolution, on that part of the lobe that lies directly over the roof of the tympanum, an area of 3 cm. in breadth was discoloured and almost black and on the verge of rupturing. Nothing abnormal was to be seen in any of the other convolutions except in the hippocampal convolution of the left side, which was much swollen and displaced.

The brain having been hardened in 10 per cent. formalin solution, was then divided into five sections after Hamilton's method. The relations and extent of the abscess could now be accurately determined. There were found to be two distinct abscess cavities; one, the smaller, of very recent formation and higher placed, was situated mainly in the lower part of the first temporal convolution. It was from this abscess that the pus had escaped on removing the brain. The lower abscess, or the primary abscess, was confined chiefly to the middle and lower temporal convolutions, measured 50 mm. from before backwards, 40 mm. laterally, and 25 mm. from above downward and held over 40 cc. of fluid. This abscess had a firm and thick capsule with a smooth inner surface. There was no communication between the two abscesses, or between either of them and the ventricles. The walls of the upper abscess were irregular, the tissue being necrotic and discoloured. There was no trace of membrane. This abscess, as already mentioned, corresponded to the lower part of the first temporal convolution, extending internally from a depth of about 3 mm. from the cortex, reaching in front close to the external capsule near the base and anterior end of the lenticular nucleus, and behind to the level of the descending horn of the lateral ventricle.

From examination of the different sections of the brain an idea of the extent and relations of the abscess can be made out.

Section I.—Brain tissue normal. This section was composed of Broca's middle and superior frontal convolutions.

Section II.—Anterior surface: no disease can be seen. Posterior surface: superiorly a distinct necrotic area of the secondary or superior abscess is clearly seen lying in the substance of the superior temporo-sphenoidal convolution below the well marked fissure of Sylvius, extending from near the cortex inwards to the external and inferior angle of the lenticular nucleus. Below can be seen the capsular lining of the anterior end of the lower abscess cavity. The white fibres arising from this temporo-sphenoidal lobe and going to join the fibres of the internal capsule, are markedly destroyed. On handling this section we can make out that the disease is distinctly confined to the temporo-sphenoidal convolutions. The fissure of Sylvius intervenes between the diseased convolutions of the lobe and the precentral and postcentral convolutions above, and Broca's convolution in front.

Section III.—Here again the disease is distinctly confined to the temporo-sphenoidal convolutions. Posteriorly we see two large abscess cavities bounded above by the fissure of Sylvius, internally by the descending horn of the lateral ventricle, and below by the swollen hippocampal convolution, the lingual and the fusiform. On the under surface of this section, near the external edge of the inferior temporo-sphenoidal convolution, is a necrotic area, the most dependent part of the large abscess cavity, and the wall here is very thin and on the verge of rupturing. On holding the section up to the light this is easily demonstrated.

Section IV.—Anterior surface: here again can be noticed the posterior extremities of the two abscess cavities bounded laterally and above by the hinder quarter of the Sylvian fissure, and internally by the posterior and descending horns of the lateral ventricle; the posterior surface shows no trace of disease.

After the examination of the five sections one is persuaded that the disease was strictly confined to the temporo-sphenoidal convolutions, and that the angular gyrus, supramarginal and frontal convolutions were intact.

In both cases the type of aphasia was the same, a loss of the power of naming objects. In both cases there was not sufficient visual defect from the optic neuritis to interfere with fairly good vision, neither was there any cortical visual defect as far as could be ascertained. Both patients recognised fully the use of objects presented to them; they also understood without difficulty spoken language, showing that there was no word deafness.

The examination of the brain after death in the second case, showed that neither the posterior part of the first temporal, nor the third left frontal convolution was interfered with, the destruction of tissue being confined to the lower and more anterior parts of the second and third temporal convolutions. Although no examination of the brain in the first case was obtained, it is almost certain that the destruction of tissue occurred in the same area as the other case, that is, it was confined to the lower and anterior part of the temporal convolution. The rapid and almost complete recovery of the power of speech after the operation is strong evidence in support of the limitation of the destructive process to the temporal lobe.

How is an aphasia solely characterised by an inability to name things to be explained by a temporal lobe lesion? Is it due to destruction of what has been called the *naming centre*? There is considerable evidence in favour of setting aside a special part of the brain for such a centre. It was first suggested by Broadbent, and more recently ably supported from the results of clinical observations by Dr. C. K. Mills of Philadelphia. He looks upon the third temporal as the seat of the naming centre. The inability to name objects may, however, be due to the cutting off of the association tracts between the visual centre and the auditory centre, on the one side, and the latter and the motor centre for speech, on the other.

Whether these two cases were examples of direct injury to a "naming centre" or the cutting off of it from its connections, I am unable to say, but they are both of value, especially the second, as showing that a form of aphasia characterised by an inability to remember the name of objects or to recollect names, may arise from a destructive lesion confined to the temporo-sphenoidal lobe. This form of aphasia, present in a any suspected case of brain abscess, would, therefore, be of value in determining its localization.





STEWART—APHASIA.

To illustrate the conditions found in Case No. II.

FIG. I.

THIRD VERTICAL SECTION.—ANTERIOR SURFACE.

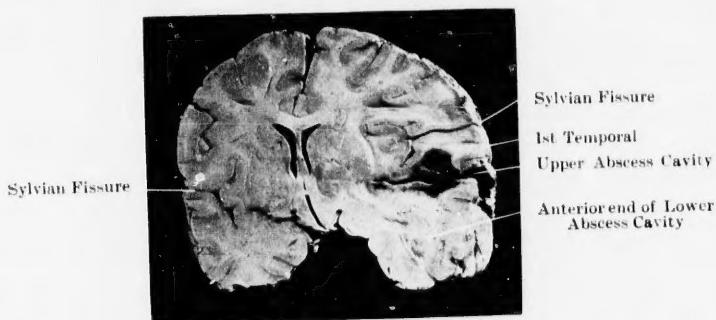
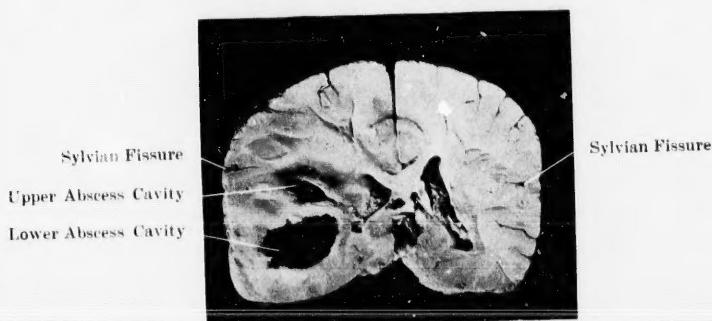


FIG. II.

FOURTH VERTICAL SECTION.—POSTERIOR SURFACE.

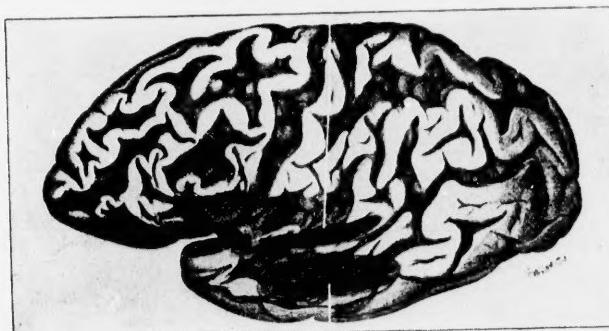


STEWART—APHASIA.

FIG. III.

Showing outline on the cortex corresponding to the abscess cavities.

Outline of Upper Abscess Cavity.



Outline of Lower Abscess Cavity.

BIBLIOTHÈQUE  
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